CHAPTER 19

# Respiratory Rate and Abnormal Breathing Patterns

## KEY TEACHING POINTS

- Respirations should be observed for at least 60 seconds, not only to increase the detection of tachypnea but also to uncover unusual breathing patterns, such as Cheyne-Stokes breathing.
- Tachypnea is a valuable diagnostic and prognostic sign in a variety of conditions. In patients with altered mental status, bradypnea (≤12 breaths/minute) increases the probability of opiate intoxication.
- In hospitalized patients, Cheyne-Stokes breathing is an accurate sign of left ventricular dysfunction, especially in patients aged ≤80 years. It is present in one out of three patients with reduced ejection fraction.
- Abnormal respiratory abdominal movements—abdominal paradox and asynchronous breathing—are best observed when the patient is supine. These signs indicate respiratory muscle weakness, either diaphragm paralysis (abdominal paradox) or a patient who is tiring from the distress of bronchospasm (asynchronous breathing).
- Orthopnea, trepopnea, and platypnea describe tachypnea that appears only in particular patient positions. Each has specific diagnostic significance.

## RESPIRATORY RATE

### I. INTRODUCTION

The respiratory rate (i.e., number of breaths per minute) is one of the four traditional vital signs, the others being heart rate, blood pressure, and temperature. One of the first clinicians to recommend routine measurement of the respiratory rate was Stokes in 1825,[1] although routine charting of this vital sign was infrequent until the late 19th century.[2,3]

### II. TECHNIQUE

The respiratory rate is usually measured while the clinician is holding the patient's wrist and ostensibly measuring the pulse, primarily because the respiratory rate may change if attention is drawn to it. This practice seems reasonable, because the respiratory rate is the only vital sign under voluntary control.

As routinely recorded in the patient's hospital record, the respiratory rate is often inaccurate.[4,5] In studies of patients whose actual respiratory rates ranged from 10 to more than 30 breaths/minute, the recorded rates clustered around 16 to 22 breaths/minute 75% to 98% of the time.[5,6] These errors usually reflect too

short a period of observation (i.e., the clinician counting the number of breaths in 15 seconds, multiplying the result times 4); in one study, 15 seconds of observation detected only 23% of tachypneic patients, whereas 60 seconds of observation detected every tachypneic patient.[6] Consequently, respirations should be observed for at least 60 seconds, not only to increase accuracy of the measured rate but also to allow for the detection of unusual breathing patterns, such as Cheyne-Stokes respirations (see later).

# III. FINDING

## A. THE NORMAL RESPIRATORY RATE

The normal respiratory rate averages 20 breaths per minute (range 16 to 25 breaths/minute), based on careful measurement in persons without fever, heart disease, or lung disease.[7,8] This estimate is identical to that made over 150 years ago by Lambert Quetelet, who was the first to compile and analyze vital and social statistics.*[9] For unclear reasons, many textbooks, citing no data, mistakenly record the normal rate as 12 to 18 breaths/minute.[7]

## B. TACHYPNEA

Definitions of tachypnea vary, but the most commonly applied definition, based on the normal range and clinical studies, is respirations of 25 breaths/minute or more.

## C. BRADYPNEA

Bradypnea is variably defined as respiratory rates less than 8 to 12 breaths/minute. In patients receiving epidural opiate analgesia, respiratory rates less than 8 to 10/minute are the best definition of respiratory depression, a finding heralding respiratory failure.[10] In patients with altered mental status who are evaluated by medics, a respiratory rate of 12/minute or less best identifies those intoxicated with opiates. (See the section on Clinical Significance.)[11]

# IV. CLINICAL SIGNIFICANCE

## A. TACHYPNEA

The finding of tachypnea has both diagnostic and prognostic value. As a diagnostic sign, tachypnea argues modestly for the diagnosis of pneumonia in outpatients with cough and fever (likelihood ratio [LR] = 2.7; EBM Box 19.1). Tachypnea also increases the probability of pneumonia in hospitalized patients, the abnormal sign sometimes appearing as early as 1 to 2 days before the diagnosis is apparent by other means.[8,24] In patients with pneumatosis intestinalis (i.e., small cysts of gas in the bowel wall on radiologic images), tachypnea increases the probability that the surgeon will find ischemia or obstruction at laparotomy (LR =16.4).†

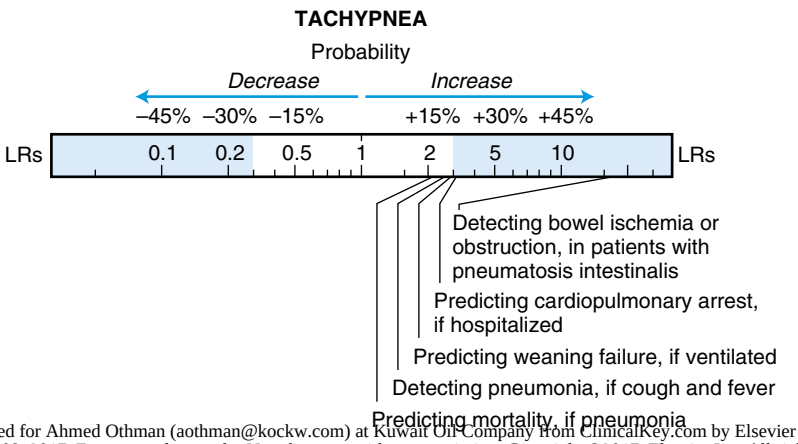
*Quetelet's 1835 monumental treatise also provided our current formula for body mass index, known as the Quetelet index (see Chapter 13).

†In these patients, tachypnea is more accurate than other CT findings, such as portal venous gas (LR = 4), dilated loops of bowel (LR NS), or pneumoperitoneum (LR NS). In this study, 52% of patients with pneumatosis intestinalis had bowel ischemia or obstruction; 48% had more benign etiologies.

## EBM BOX 19.1
*Tachypnea**

| Finding (Reference) | Sensitivity (%) | Specificity (%) | Likelihood Ratio† if Finding Is | |
|---|---|---|---|---|
| | | | *Present* | *Absent* |
| ***Rate >20/min*** | | | | |
| Detecting operative finding of intestinal ischemia or obstruction in patients with pneumatosis intestinalis[12] | 27 | 98 | **16.4** | 0.7 |
| ***Rate >24/min*** | | | | |
| Predicting failure of weaning from the ventilator, in intubated patients[13] | 94 | 68 | 2.9 | NS |
| ***Rate >27/min*** | | | | |
| Predicting cardiopulmonary arrest in medical inpatients[14] | 54 | 82 | **3.1** | 0.6 |
| ***Rate >28/min*** | | | | |
| Detecting pneumonia in patients with cough and fever[15-18] | 7-36 | 80-99 | 2.7 | 0.9 |
| ***Rate >30/min*** | | | | |
| Predicting hospital mortality in patients with pneumonia[19-23] | 41-85 | 63-87 | 2.1 | 0.6 |

*Diagnostic standard: For *failure of weaning,* progressive hypoxemia or respiratory acidosis; for *pneumonia,* infiltrate on chest radiograph.
†Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
*NS,* Not significant.
Click here to access calculator



One characteristic of a vital sign is that it accurately predicts the patient's prognosis, and EBM Box 19.1 shows that tachypnea predicts subsequent cardiopulmonary arrest in hospitalized patients (LR = 3.1) much better than tachycardia or abnormal blood pressure.[14] During trials of weaning from a ventilator, tachypnea is a significant though modest predictor of weaning failure (LR = 2.9).[13,25] In patients hospitalized with pneumonia, severe tachypnea (i.e., rate >30 breaths/minute) predicts subsequent hospital death (LR = 2.1).

### B. TACHYPNEA AND OXYGEN SATURATION

The respiratory rate correlates poorly with the patient's level of oxygen desaturation ($r$ = 0.16).[26] Although this initially seems surprising (i.e., the lower the oxygen level, the more rapid a patient should breathe), this actually is expected because some hypoxemic patients, by breathing rapidly, are able to bring their oxygen level back up to normal (i.e., hyperventilation increases arterial oxygen levels) and because other patients are hypoxemic simply because they have a primary hypoventilatory disorder. Consequently, the respiratory rate and oxygen saturation are both valuable to the clinician, each providing information independent from the other.

### C. BRADYPNEA

In a study of patients seen by medics for altered mental status, the finding of a respiratory rate of 12 or less predicted a positive response to naloxone, thus confirming the clinical impression of opiate intoxication (sensitivity of 80%, specificity of 95%, positive LR = 15.5, and negative LR = 0.2).[11]

# ABNORMAL BREATHING PATTERNS

## I. CHEYNE-STOKES BREATHING (PERIODIC BREATHING)

### A. INTRODUCTION

Cheyne-Stokes breathing consists of alternating periods of apnea and hyperpnea (Fig. 19.1). Some authors equate the term **periodic breathing** with Cheyne-Stokes breathing,[27,28] while others reserve periodic breathing to oscillations of tidal volume that lack intervening periods of apnea.[29]

Cheyne-Stokes breathing was described by John Cheyne in 1818 and William Stokes in 1854.[30]

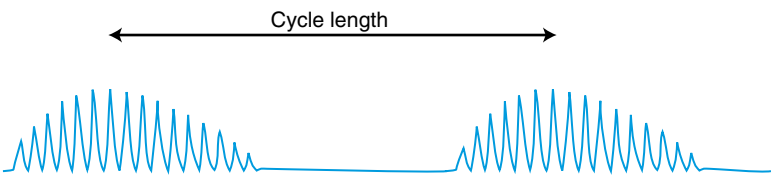



**FIG. 19.1 CHEYNE-STOKES RESPIRATION.** There are alternating cycles of hyperpnea and apnea. During the hyperpnea phase, only the tidal volume oscillates; the respiratory frequency is constant.

## B. THE FINDING

### 1. THE BREATHING PATTERN

At the end of each apneic period, breathing commences with excursions of the chest that initially are small but gradually increase for several breaths and then diminish until apnea returns. The respiratory rate is constant during the hyperpnea phase and does not gradually increase and then decrease as often surmised.[31] Cheyne-Stokes breathing often first appears when the patient lies down, probably because this position reduces the patient's functional residual capacity, thus diminishing the lung's ability to buffer changes in carbon dioxide.[28,32] (See the section on Pathogenesis later.)

The time between two consecutive peaks of hyperpnea is called the **cycle length** or **period**. Each cycle length is divided into a hyperpnea phase (lasting about 30 seconds on average in patients with congestive heart failure) and an apnea phase (lasting about 25 seconds on average).[33,34]

### 2. ASSOCIATED BEDSIDE OBSERVATIONS

Several additional findings appear in patients with Cheyne-Stokes breathing. During the hyperpnea phase, the patient is alert and sometimes agitated, with dilated pupils, hyperactive muscle stretch reflexes, and increased muscle tone. During the apnea phase, the patient appears motionless and asleep with constricted pupils, hypoactive reflexes, and reduced muscle tone.[35,36] The agitation of the hyperpnea phase can easily startle a patient out of sleep, a symptom that clinicians can mistake for the paroxysmal nocturnal dyspnea of heart failure caused by transient pulmonary edema.[37,38]

## C. CLINICAL SIGNIFICANCE

### 1. ASSOCIATED CONDITIONS

Cheyne-Stokes breathing affects 30% of patients with stable congestive heart failure.[29,34] The breathing pattern also appears in many neurologic disorders, including hemorrhage, infarction, tumors, meningitis, and head trauma involving the brainstem or higher levels of the central nervous system.[35,39] Normal persons often develop Cheyne-Stokes breathing during sleep[27] or at high altitudes.[35]

In patients hospitalized on an inpatient medicine service, the finding of Cheyne-Stokes respirations increases the probability of left ventricular systolic dysfunction (i.e., ejection fraction less than 40%; LR = 5.4; EBM Box 19.2). The finding is more accurate in patients under the age of 80 years (LR = 8.1) than in patients over the age of 80 years (LR = 2.7), suggesting that alternative explanations of Cheyne-Stokes breathing (e.g., central nervous system injury) are more important in older patients.[34]

### 2. PROGNOSTIC IMPORTANCE

Although Dr. Stokes originally believed that Cheyne-Stokes respirations implied a poor prognosis in patients with heart failure, modern studies demonstrate contradictory results, some showing that the finding implies worse survival,[40] while others show no association with increased mortality.[34]

## D. PATHOGENESIS

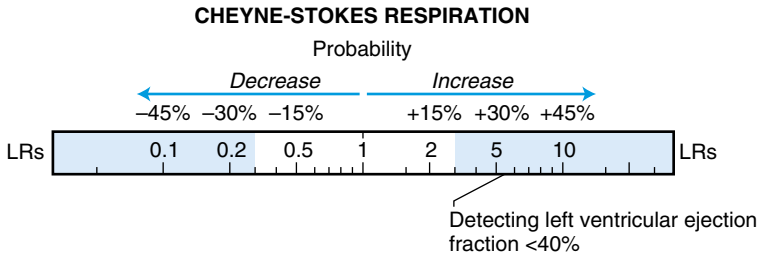
The fundamental problem causing Cheyne-Stokes breathing is enhanced sensitivity to carbon dioxide. The circulatory delay between the lungs and systemic arteries, caused by poor cardiac output, also contributes to the waxing and waning of breaths. Cerebral blood flow increases during hyperpnea and decreases during apnea, perhaps explaining the fluctuations of mental status.[33,41]

**EBM BOX 19.2**

*Cheyne-Stokes Breathing, Detecting Reduced Ejection Fraction*[34,*]

| Finding (Reference) | Sensitivity (%) | Specificity (%) | Likelihood Ratio† if Finding Is | |
|---|---|---|---|---|
| | | | *Present* | *Absent* |
| All adults | 33 | 94 | **5.4** | 0.7 |
| Patients aged ≤80 years | 32 | 96 | **8.1** | 0.7 |
| Patients aged >80 years | 42 | 84 | 2.7 | NS |

*Diagnostic standard: For *reduced ejection fraction,* <40% by transthoracic echocardiography.
†Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
*NS,* Not significant.
Click here to access calculator




## 1. ENHANCED SENSITIVITY TO CARBON DIOXIDE

Whether because of congestive heart failure or neurologic disease, patients with Cheyne-Stokes breathing have 2 to 3 times the normal sensitivity to carbon dioxide.[39,42] This causes patients to hyperventilate excessively, eventually driving the carbon dioxide level so low that central apnea results. After they stop breathing, carbon dioxide levels again rise, eliciting another hyperventilatory response and thus perpetuating the alternating cycles of apnea and hyperpnea.

Mountain climbers develop Cheyne-Stokes breathing because hypoxia induces hypersensitivity to carbon dioxide. In contrast, their native Sherpa guides, who are acclimated to hypoxia, lack an exaggerated ventilatory response and do not develop Cheyne-Stokes breathing.[35]

## 2. CIRCULATORY DELAY BETWEEN LUNGS AND ARTERIES

Ventilation is normally controlled by the medullary respiratory center, which monitors arterial carbon dioxide levels and directs the lungs to ventilate more if carbon dioxide levels are too high and less if levels are too low. The medulla signals the lungs almost immediately, the message traveling via the nervous system. The feedback to the medulla, however, is much slower because it requires circulation of the blood from the lungs back to the systemic arteries.

In Cheyne-Stokes breathing, the carbon dioxide levels in the alveoli and those of the systemic arteries are precisely out of sync. During peak hyperpnea, carbon dioxide levels in the alveoli are very low, yet the medulla is just beginning to sample blood containing high carbon dioxide levels from the previous apnea phase and

thus still directs the lungs to continue breathing deeply.[35] The delay in feedback to the medulla contributes to the gradual waxing and waning of tidal volume.

The length of circulatory delay also governs the cycle length of Cheyne-Stokes breathing, the two correlating closely ($r = 0.8$ between cycle length and circulation time from lungs to arteries, $p < 0.05$).[33,41] The cycle length is about 2 times the circulation time, just as would be expected from the observation that carbon dioxide levels in the lungs and arteries are precisely out of sync. Nonetheless, one study showed poor correlation between cycle length and ejection fraction,[34] indicating either that ejection fraction is a poor measure of circulation time or that variables other than cardiac performance govern cycle length.

# II. KUSSMAUL RESPIRATIONS

Kussmaul respirations are rapid and deep and appear in patients with metabolic acidosis.[43] The unusually deep respirations are distinctive, because other causes of tachypnea, such as heart and lung disease, reduce vital capacity and thus cause rapid, *shallow* respirations.

In children with severe malaria, the finding of Kussmaul respirations detects a severe metabolic acidosis with a sensitivity of 91%, specificity of 81%, positive LR = 4.8, and negative LR = 0.1.[44]

# III. GRUNTING RESPIRATIONS

## A. DEFINITION

Grunting respirations are short, explosive sounds of low-to-medium pitch produced by vocal cord closure during expiration. The actual sound is the rush of air that occurs when the glottis opens and suddenly allows air to escape. Grunting respirations are more common in children,[45] although the finding also has been described in adults as a sign of respiratory muscle fatigue[46] and, in the preantibiotic era, as a cardinal sign of lobar pneumonia, usually appearing after 4 to 6 days of illness.[3,47]

## B. PATHOGENESIS

Grunting respirations slow down expiration and allow more time for maximal gas exchange.[46] In animal experiments, artificial mimicking of grunting respirations causes the $PO_2$ to increase by 10% and the $PCO_2$ to fall by 11%, whether or not the animal has pneumonia.[48] Grunting respirations also produce positive pressure exhalation that may reduce exudation of fluid into the alveoli, based on an old observation that the administration of morphine to patients with pneumonia often reduced grunting respirations but was sometimes immediately followed by fatal pulmonary edema.[47]

# IV. ABNORMAL ABDOMINAL MOVEMENTS

## A. NORMAL ABDOMINAL MOVEMENTS

In the absence of massive gaseous distention, the abdominal viscera are noncompressible and act like hydraulic coupling fluid that directly transmits movements of the diaphragm to the anterior abdominal wall.[49] Abdominal respiratory movements, therefore, indirectly indicate how the diaphragm is moving. During normal respiration, the chest and abdomen move synchronously: both out during inspiration and

both in during expiration (Fig. 19.2). The chest wall moves more when the person is upright, and the abdomen moves more when the person is supine.[50,51]

## B. ABNORMAL ABDOMINAL MOVEMENTS

Three abnormal abdominal movements are all signs of chronic airflow obstruction or respiratory muscle weakness: **asynchronous breathing, respiratory alternans,** and **paradoxical abdominal movements**.

### 1. ASYNCHRONOUS BREATHING

#### A. FINDINGS

Asynchronous breathing is an abnormal *expiratory* movement that sometimes develops in patients with chronic airflow obstruction. In these patients, the normal smooth inward abdominal movement during expiration is replaced by an abrupt inward and then outward movement (see Fig. 19.2).[52,53]

#### B. CLINICAL SIGNIFICANCE

In patients with chronic airflow obstruction, asynchronous breathing correlates with lower forced expiratory volumes and a much poorer prognosis.[53] Among

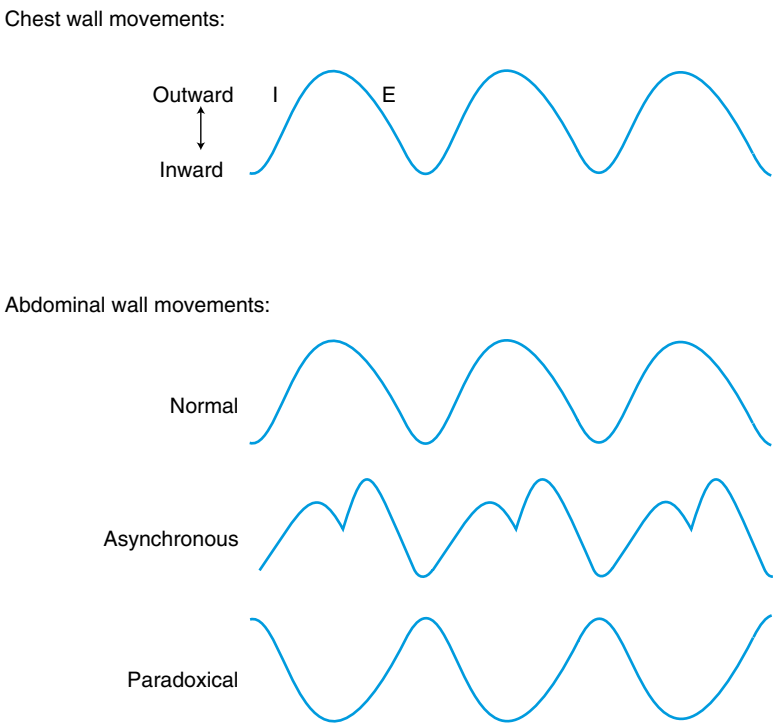



**FIG. 19.2 RESPIRATORY ABDOMINAL MOVEMENTS.** Chest movements are depicted in the first row. "I" denotes inspiration and "E" denotes expiration. Upward sloping lines on the drawing indicate outward body wall movements; downward sloping lines indicate inward movements. In normal persons, the abdominal and chest wall movements are completely in sync. In asynchronous breathing, only expiratory abdominal movements are abnormal. In paradoxical abdominal movements, both inspiratory and expiratory abdominal movements are abnormal (see text).

patients with chronic airflow obstruction who develop acute respiratory symptoms, the presence of an asynchronous breathing pattern predicts subsequent hospital death or the need for artificial ventilation with a sensitivity of 64%, specificity of 80%, and positive LR of 3.2 (negative LR not significant).[52]

#### C. PATHOGENESIS

The outward abdominal movement during expiration probably reflects the strong action of chest wall accessory muscles during expiration, which push the flattened diaphragm temporarily downward, and thus the abdomen abruptly outward.[50,52]

### 2. RESPIRATORY ALTERNANS

Respiratory alternans describes a breathing pattern that alternates between inspiratory movements that are mostly abdominal and inspiratory movements that are mostly thoracic.[25]

### 3. PARADOXICAL ABDOMINAL MOVEMENTS

#### A. FINDING

Paradoxical abdominal movements are completely out of sync with those of the chest wall. During inspiration, the abdomen moves in as the chest wall moves out; during expiration, the abdomen moves out as chest moves in.[49,54-56]

#### B. CLINICAL SIGNIFICANCE

Paradoxical abdominal movements are a sign of bilateral diaphragm weakness. Most of these patients also complain of severe orthopnea. In one study of patients with dyspnea and neuromuscular disease, the finding of paradoxical abdominal movements detected diaphragm weakness with a sensitivity of 95%, specificity of 70%, and positive LR of 3.2. (In this study, the definition of paradoxical movements was any inspiratory inward abdominal movement, and the definition of diaphragm weakness was a maximal transdiaphragmatic pressure ≤30 cm $H_2O$; the normal sniff transdiaphragmatic pressure is >98 cm $H_2O$.)[54]

#### C. PATHOGENESIS

If the diaphragm is totally paralyzed, the inspiratory outward movement of the chest wall will draw the diaphragm upward, and thus the abdomen draws inward. The weight of the abdominal viscera probably also plays a role, because paradoxical movements are most obvious in affected patients who are positioned supine and are often absent when the patient is upright.[54]

A mimic of paradoxical abdominal movements is seen in patients with tetraplegia. In these patients, respiratory motion relies entirely on the diaphragm: as it descends during inspiration, pushing the abdominal wall out, the paralyzed chest wall may be drawn inward. The chest and abdomen are completely out of sync in these patients, but in contrast to the paradoxical abdominal movements of diaphragm weakness, the abdominal wall of tetraplegia patients moves *outward* during inspiration, not *inward*.

## V. ORTHOPNEA, TREPOPNEA, AND PLATYPNEA

These terms describe tachypnea (and dyspnea) that appears abruptly in particular positions: when the patient is supine (**orthopnea**), lying on one side (**trepopnea**), or upright (**platypnea**). These findings are often first diagnosed during observation of the patient's respirations at the bedside.

## A. ORTHOPNEA

### 1. FINDING

Orthopnea describes dyspnea that appears when the patient lies down but is relieved when the patient sits up (from the Greek words *ortho* meaning straight or vertical, and *pnea* meaning to breathe).

### 2. CLINICAL SIGNIFICANCE

Orthopnea occurs in a variety of disorders, including massive ascites, bilateral diaphragm paralysis, pleural effusion, morbid obesity, and severe pneumonia, although its most important clinical association is congestive heart failure.[54,55,57] In one study of patients with known chronic obstructive pulmonary disease, the finding of orthopnea distinguished between those patients with abnormally low ejection fraction (less than 50%) and those with normal ejection fraction with a sensitivity of 97%, specificity of 64%, positive LR of 2.7, and negative LR of 0.04.[58] This suggests that in patients with lung disease, the *presence* of orthopnea has limited value (i.e., occurs in both lung and heart disease), but the *absence* of orthopnea is more compelling, *decreasing* the probability of associated left ventricular dysfunction (LR = 0.04).

### 3. PATHOGENESIS

In patients with orthopnea, lung compliance and vital capacity decrease significantly after moving from the upright to supine position. This explains in part why dyspnea worsens in the supine position and why orthopnea is a finding common to so many different clinical conditions.[57,59,60] Nonetheless, orthopnea cannot be entirely caused by postural changes in lung mechanics, for several reasons. First, orthopnea is uncommon in other disorders with similar reductions of vital capacity and compliance (e.g., interstitial fibrosis). Second, in patients with congestive heart failure, orthopnea correlates poorly with the pulmonary artery wedge pressure, which should have some relation to interstitial edema and pulmonary mechanics.[61] Finally, elevation of the head alone brings prompt relief to some orthopneic patients. It was once believed that elevation of the head relieved dyspnea because it reduced intracranial venous pressure and thus improved cerebral perfusion, although this hypothesis has been experimentally disproved.[57]

## B. TREPOPNEA

### 1. FINDING

Trepopnea[‡] (from Greek *trepo* meaning twist or turn) describes dyspnea that is worse in one lateral decubitus position and relieved in the other.

### 2. CLINICAL SIGNIFICANCE

There are three principal causes of trepopnea.

#### A. UNILATERAL PARENCHYMAL LUNG DISEASE[64,65]

Affected patients usually prefer to position their healthy lung down, which improves oxygenation because blood preferentially flows to the lower lung.

[‡] In 1937, Drs. Wood and Wolferth first described trepopnea in patients with congestive heart failure.[62] In searching for a name for the finding, a patent lawyer suggested to them *rolling relief*, which they translated into *rotopnea*, until a Dr. Kern pointed out that *roto* was a Latin root and the pure Greek term *trepopnea* would be better (Wood, 1959).[63]

### B. CONGESTIVE HEART FAILURE FROM DILATED CARDIOMYOPATHY[62,63,66]

Patients usually prefer to have their right side down. Whether this is due to positional changes in lung mechanics (e.g., left lung atelectasis from cardiomegaly), right ventricular preload, or airway compression is unclear. The preference for the right side down in cases of heart failure may contribute to the right-sided predilection of pleural effusions in these patients.[67]

### C. MEDIASTINAL OR ENDOBRONCHIAL TUMOR

Tumors may compress the airways or central blood vessels in one position but not the other.[68-70] A clue to this diagnosis is a localized wheeze that appears in the position causing symptoms.[68]

### D. OTHER CAUSES

Rare reports of trepopnea include a patient with position-dependent right-to-left intracardiac shunting[71] and a patient with unilateral diaphragmatic paralysis.[72] The patient with hemidiaphragm paralysis (on the right side) had left-sided trepopnea, possibly because this position increased the weight of abdominal viscera against the only functioning half of the diaphragm.[72]

## C. PLATYPNEA

### 1. FINDING

Platypnea (from the Greek *platus*, meaning "flat") is the opposite of orthopnea: Patients experience worse dyspnea when upright (sitting or standing) and relief after lying down. (A related term, *orthodeoxia*, describes a similar deterioration of oxygen saturation in the upright position.) This rare syndrome was first described in 1949, and the term *platypnea* was first coined in 1969.[73,74]

### 2. CLINICAL SIGNIFICANCE

Platypnea occurs in patients with right-to-left shunting of blood through intracardiac or intrapulmonary shunts.

### A. RIGHT-TO-LEFT SHUNTING OF BLOOD THROUGH A PATENT FORAMEN OVALE OR ATRIAL SEPTAL DEFECT

These patients often first develop the finding after undergoing pneumonectomy or developing a pulmonary embolus or pericardial effusion, which for unclear reasons promotes right-to-left shunting in the upright position.[75-80]

### B. RIGHT-TO-LEFT SHUNTING OF BLOOD THROUGH INTRAPULMONARY SHUNTS

Right-to-left shunting of blood through intrapulmonary shunts located in the bases of the lungs occurs in the hepatopulmonary syndrome, a complication of chronic liver disease (see Chapter 8)[81] and hereditary hemorrhagic telangiectasia.[82] In these patients, the upright position causes more blood to flow to the bases, thus aggravating the right-to-left shunting of blood and the patient's hypoxemia.

*The references for this chapter can be found on www.expertconsult.com.*

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